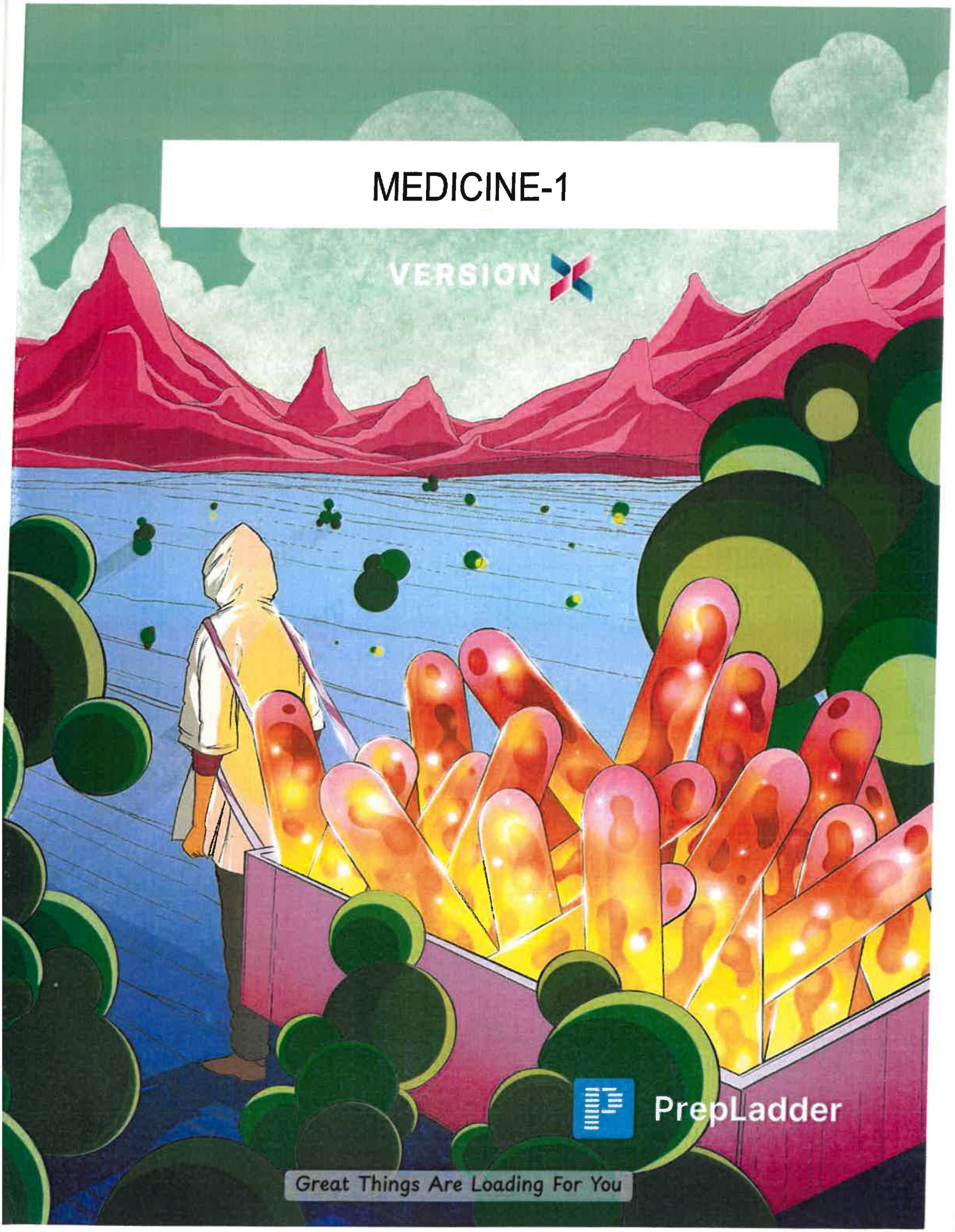
MEDICINE-1

VERSION X



PrepLadder

Great Things Are Loading For You

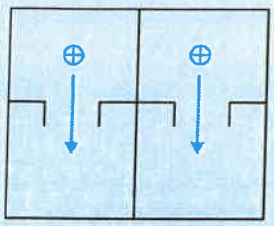
1. HEART SOUNDS 1

HEART SOUNDS

CARDIAC CYCLE

00:00:45

ATRIAL SYSTOLE

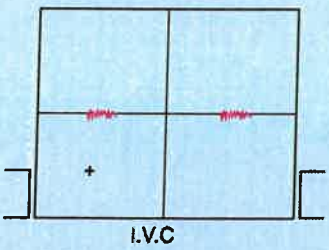


Atrial systole 30% filling

- Atria contracts → filling of ventricles begins
- Diastole → 70% of ventricular filling
- Atrial systole → 30% of ventricular filling
- In Atrial fibrillation → ↓ Atrial contraction →↓ filling of ventricles → ↓ Output → Hypotension
  - Complication - Clots → embolism → Acute ischemic stroke

ISOVOLUMETRIC CONTRACTION (IVC)

$S_1 = M_1\ T_{1c}$

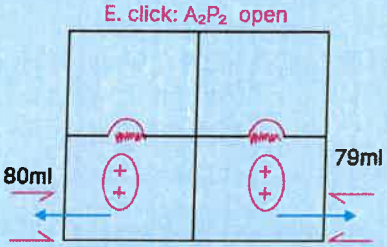


I.V.C

- Closure of tricuspid & mitral valve → 1st heart sound / S_1 (at end of atrial systole /at start of Isovolumetric contraction)
  - $S1 = M1\ T1_c$
- No blood comes in and goes out from ventricles → Isovolumetric contraction

VENTRICULAR SYSTOLE

E. click: A_2P_2 open

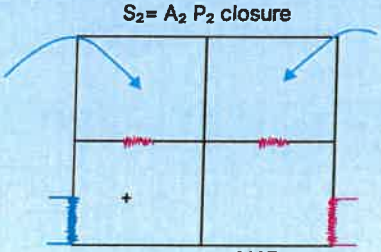


V.systole

- Ventricular pressure starts building up
  - Slight bulge in mitral valve & tricuspid valve
  - Both ventricles contract with equivalent power (at same time)
- 80 ml blood pumped to aorta & pulmonary artery
- Opening of aortic and pulmonary valves (A_2P_2) (during ventricular systole) → ejection click
- Example - Damage to left ventricle → 79 ml pumped to aorta → Pooling of blood → Pulmonary edema

ISOVOLUMETRIC RELAXATION (IVR)

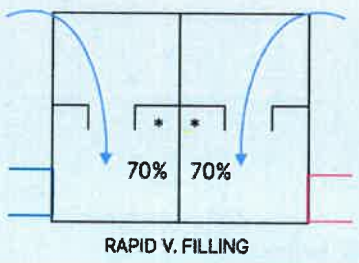
$S_2 = A_2\ P_2$ closure



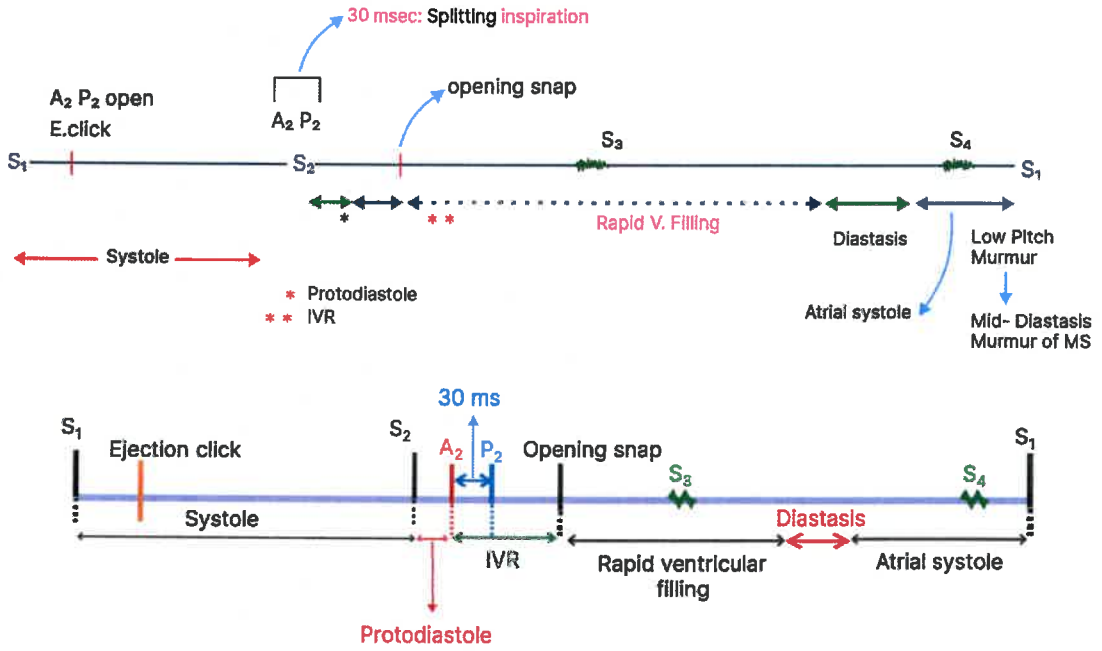
I.V.R

- Aortic and pulmonary (A_2P_2) valve closure → 2nd Heart sound S_2 (at end of ventricular systole)
  - S2 - A2P2
- No bulge in mitral valve & tricuspid valve
  - Cause - Decompression of ventricles
- All valves (mitral/tricuspid & aortic/pulmonary) closed → heart relaxes (isovolumetric relaxation)

STAGE OF RAPID VENTRICULAR FILLING



- Flow of blood directly into ventricles
  - 70% filling of blood in ventricles occurs
- Opening of mitral and tricuspid (M_1T_1) valves → Opening snap
  - Loud opening snap → Mitral stenosis / Tricuspid stenosis

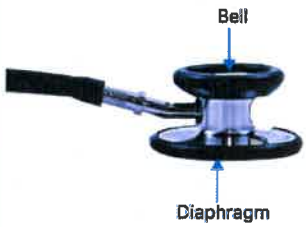


- S1 - S2 - Systole
  - Ejection click heard
- Systole is over --> Time lag of 30 msec between closure of Aortic valves (1st closes) & pulmonic valve
  - Splitting of 2nd Heart sound / S2 → More prominent on inspiration

- S2 - S1 - Diastole → Divided to -
  - Protodiastole - End of ventricular systole to Closure of Aortic & pulmonic valves
  - Isovolumetric relaxation - Closure of Aortic & pulmonic valves to opening of tricuspid & mitral valves
  - Rapid ventricular filling - 70% ventricular filling of blood
  - Diastasis - 'Heart stands stills' physiologically --> least cardiac activity
  - Atrial systole - Terminal part of diastole (when ventricles relax before S1)

- 3rd heart sound / S3 / Ventricular gallop rhythm
  - Occurs during rapid ventricular filling
  - < 35 years of age - heard physiologically
  - >35 years of age - heard on pathology (Eg - CHF)

- 4th heart sound / S4 / Atrial gallop rhythm
  - Occurs during atrial systole
  - Cause - Pathology (All age groups)

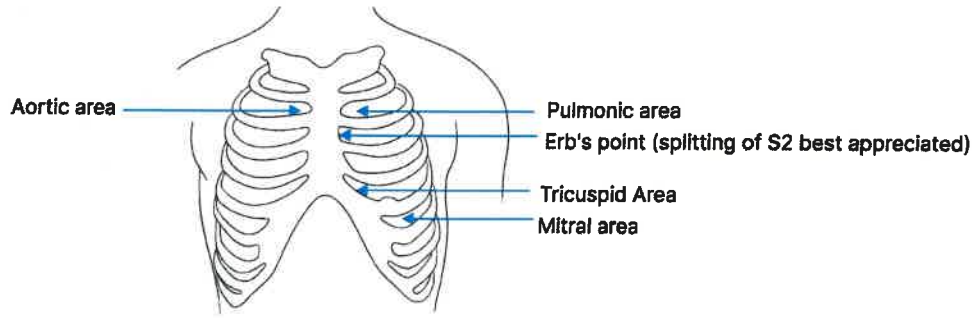


Important Information

- ↑ pitch heart sounds → heard through diaphragm (of stethoscope)
  - S1, S2, Opening snap
- ↓ pitch heart sounds → heard through bell (of stethoscope)
  - S_3, S_4
  - Tumor plop sound (Seen on benign cardiac tumors (Eg - Atrial myxoma)
- ↓ pitch/mid-diastolic murmur (heard through bell) → Mitral stenosis
  - Not involved in ↓ pitch heart sounds

CARDIAC AUSCULATION AREAS

00:22:17



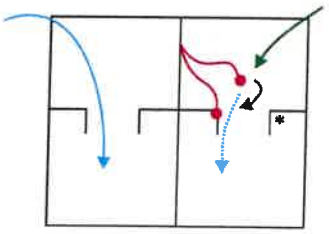
AREA	LOCATION
Aortic area	Right of sternum at 2nd Intercostal space
Pulmonic area	Left of sternum at 2nd Intercostal space
Erb's point (splitting of S2 best appreciated)	Left of sternum at 3rd Intercostal space
Tricuspid Area	Left of sternum at 4th Intercostal space
Mitral area	Left of sternum at 5th Intercostal space (at midclavicular line)

ATRIAL MYXOMA

00:24:45

- Pedunculated tumor
- Has stalk→ Suspended in lumen of left atrium

Blood enters left atrium
↓
Tumor acts as obstacle
↓
Blood hits tumor
↓
Tumor swings like pendulum
↓
Tumor hits open mitral valve leaflet
↓
Produces tumor plop sound



- Limited blood supply to left ventricle → Effort intolerance
- Big sized tumor → murmur of mitral stenosis (mid-diastolic murmur) heard

CLINICAL SCENARIO

- A young female presents with

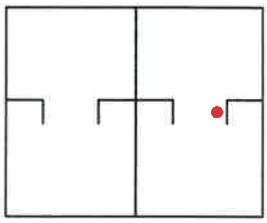
CLINICAL FEATURES	• Effort intolerance
	• Dyspnea on exertion
	• Platypnea (breathlessness while sitting → resolves on lying down)
	  o Lying down → tumor moves away from valve →↓ obstruction
	• Transient Ischemic Attacks
	  o Cause -
	  → Platelet plugs on damaged valve endothelium → embolism
	  → Tumor cell embolisation

ON EXAMINATION	• Pallor +/- • Tumor plop sound (can be missed) • Mid diastolic murmur • (Long-term) Damage to mitral leaflets - Mitral Regurgitation
CLINICAL COURSE	Repeated impact by tumor → endothelial damage of mitral valve ↓ Formation of platelet plaque ↓ Tumor cells embolisation/Platelet plaque can get dislodged ↓ Embolization → Dissemination into systemic circulation ↓ Neurological manifestations/Transient Ischemic Attack
INVESTIGATION	• IOC - Transthoracic echocardiography • Transesophageal echocardiography
MANAGEMENT	• T/t - Cardiothoracic Vascular Surgery o Surgical resection of tumor

PAPILLARY ELASTOMA

00:40:03

- Tumor originates from heart valve tissue
- Investigations -
  - o Transthoracic echocardiography / Transesophageal echocardiography
  - o Cardiac MRI
- T/t - Prosthetic valve



Important Information

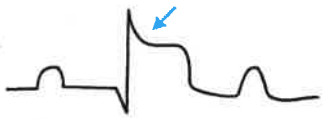
- M/c tumor of heart → Metastasis
  - Arising from Oat cell lung carcinoma
- M/c primary malignant tumor of heart → Angiosarcoma
- M/c primary malignant tumor of heart (children) → Rhabdomyosarcoma
- M/c benign tumor of heart → atrial myxoma
- M/c site of extrapulmonary tuberculosis → Cervical lymphadenopathy
  - Tonsil - Ghon focus

TUBERCULAR PERICARDITIS

00:43:35

- Constrictive pericarditis seen
- Tubercular pericarditis → Form of extrapulmonary tuberculosis

CLINICAL FEATURES		• Chest pain at rest
		  ○ Inflammation of outermost layer of heart
		• Pain radiating to left shoulder
		  ○ Relief of pain with sitting
		• Pericardial friction rub
		• ↓ grade fever/night sweats/involuntary weight loss (5% weight loss in 6 months), ↓ in appetite, cough

WORK UP	**ECG- ST ELEVATION**	**Pericarditis** • ST elevation → concave shape in upward direction • Present in all leads except - ○ Lead aVR/V1 → shows ST depression 
	ECHOCARDIOGRAPHY	• Pericardial effusion +/-
	CB-NAAT	• Sputum tested for TB

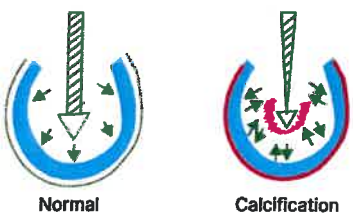
Important Information

- ECG finding in MI -
  - ST elevation has convex shape in an upward direction with T wave inversion (Pardee sign)
  - Present only in few leads

CONSTRICTIVE PERICARDITIS

00:53:17

- Development of calcification in outermost layer of heart (sequelae of tubercular pericarditis)
  - After Antitubercular therapy
- Amount of blood entering ventricles is less (<130 ml)
  - Cause - Non-compliance of heart



- Blood entering bounces off wall of ventricles → creates turbulence in ventricular cavity
- Produces cardiac auscultatory finding → Pericardial shudder/knock/shock
  - ↑ pitched heart sound, Loud diastolic sound

Important Information

- Mid systolic click → mitral valve prolapse (MVP)
  - Cause - Stretching of chorda tendinae

MCQ's

Q. Patient presents with dyspnea & distended neck veins. On auscultation, loud diastolic sound is heard with rise of JVP on inspiration. What is the diagnosis?

a. Constrictive pericarditis
b. Aortic regurgitation
c. Mitral stenosis
d. Pulmonary arterial hypertension

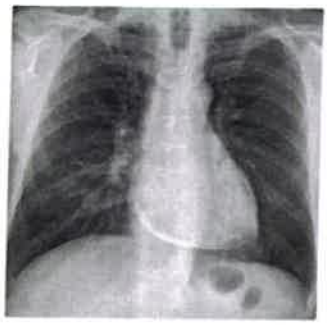
Ans: (a)

Q. Which of the following heart sound is always pathological?

a. Loud S1
b. Variable split S2
c. S3
d. S4

Ans: (d)

Q. 65-year-old woman comes to the OPD with symptoms of breathlessness & fatigue. She has undergone breast cancer treatment, including chemoradiation. On examination, engorged neck veins are noted with rapid x & y descent in JVP. CXR is shown below. Diagnosis is?



a. Constrictive pericarditis
b. Cardiac tamponade
c. Dilated cardiomyopathy
d. Restrictive cardiomyopathy

Ans: (a)

2. HEART SOUNDS PART 2

SECOND HEART SOUND / S2

00:00:58

→ Produced by

- Closure of the aortic (A2) & pulmonic (P2) valves
- Gap between the 2 closure: splitting of S2

→ P2 position is not constant : Varies with respiration

- Between A2 and inspiratory position of P2: Physiological splitting of S2 (30 msec)
  - Late P2 on inspiration
  - Early P2 on expiration
- Best appreciated by stethoscope on Erb's point
  - Left side of chest
  - 3rd intercoastal space on parasternal line
  - Splitting is best auscultated in the phase of inspiration

AORTIC STENOSIS (AS)

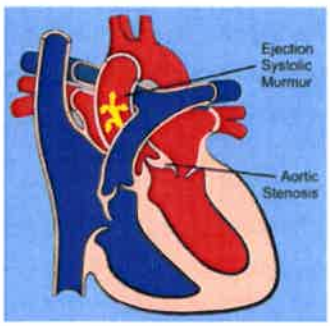
00:03:28

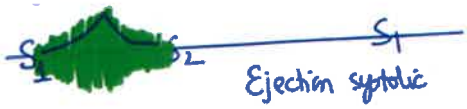
GENERAL	• Normal size of orifice of aortic valve: 3- 4 cm^2
	• Severe Aortic Stenosis: < 1 cm^2
	• Exit of blood (from heart) → takes extra time
	  ○ A2 closure : delayed closure
	  ○ **Cause**: Narrow aortic valve

CAUSES OF VALVULAR AS	• Bicuspid aortic valve (Infants) • Rheumatic fever • Calcification of valve (> 65 years)

CLINICAL FINDINGS (MNEMONIC: SAD)	• Syncope on exertion
	• Angina:
	  ○ **Cause**: Aortic Stenosis causes Left ventricular hypertrophy → ↑ O_2 demand
	• Dyspnea on exertion:
	  ○ **Cause**: Left ventricular end-diastolic pressure ↑→ Pooling of blood

EXAMINATION FINDINGS	• **Pulse**: Pulsus parvus et tardus / anacrotic pulse ○ Slow rising pulse (less amplitude)



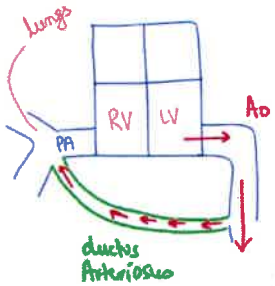
	• Heaving Apex beat
	  o **Cause**: Left-sided hypertrophy of heart
	  o Double apical impulse seen
	  o Displacement of apex beat to 6th intercoastal space heard
	• S_2:
	  o Narrow split S2
	  o A2P2 gap: < 30 msec
	  o Single S2
	  o A2 & P2 closure at same point of time (due to worsening of AS)
	  o Reverse split S2
	  o A2 closure after P2 closure (worsening of AS)
	• S_4: Atrial gallop rhythm (heard with bell)
	• Ejection systolic murmur / crescendo- decrescendo murmur heard
	 
	• Presence of Carotid thrill

IOC	• TTE (Transthoracic echocardiography)
TREATMENT	• Transcatheter Aortic Valve Implantation (TAVI)

PATENT DUCTUS ARTERIOSUS (PDA)

00:18:58

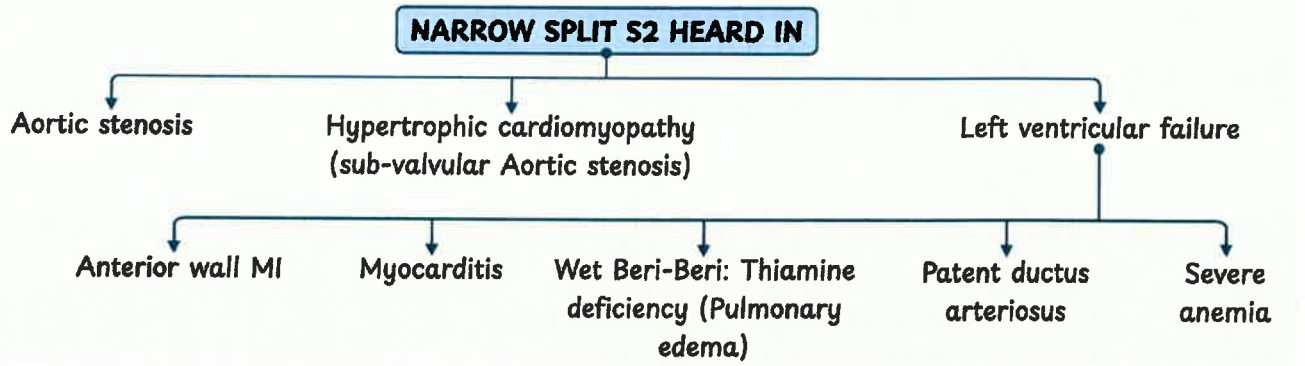
GENERAL	• Originates from bifurcation of pulmonary artery • Ends at starting of descending aorta • Begins to close at 10 – 15 hours after birth o Functional closure by Day 7 of life

PATHOPHYSIOLOGY	• Pathology: patency is maintained
	• Left → right shunt
	  o Develops Left ventricular failure
	  o Not right ventricular failure
	• Volume overloading of the left side of heart: Left ventricular failure
	  o Blood pumps from aorta to pulmonary artery (via ductus arteriosus) → Lungs (Pulmonary edema) → Left side of heart
	 

ON EXAMINATION	• S_2: Narrow split
	  o Blood exit: requires extra time
	  o Delayed A2 closure
	• Machinery/ continuous murmur
	  o Heard in systole & diastole
	  o Peaks at S2
	 

CAUSES	• Preterm baby: Hyaline Membrane Disease ○ Causing Hypoxia → produces PGE_2 → patent ductus arteriosus • Term baby: Congenital rubella syndrome
CLINICAL FEATURES	• Poor attachment to breast , Irritable • Dyspnea on breastfeeding
IOC	• TTE (Transthoracic echocardiography)
TREATMENT	• Preterm: Ibuprofen / Indomethacin IV • Term: Surgical ligation
COMPLICATIONS	• Pulmonary Artery Hypertension • Necrotizing Enterocolitis • Congestive Heart Failure • Acute Kidney Injury

Important Information

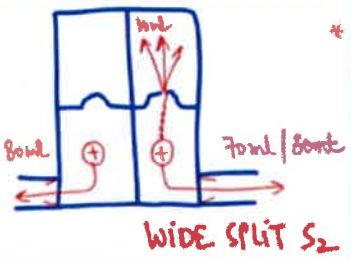


MITRAL VALVE REGURGITATION

00:36:04

CAUSES	• Mitral Valve Prolapse • Infective Endocarditis • Myocardial Ischemia
PATHOPHYSIOLOGY	• Blood leaks to left atria from left ventricle (due to defective mitral valves) (Eg:- 10 ml leak to left atria) ○ Blood shunting from left ventricle to left • Less blood pumped to aorta (Eg:- 70 ml pumped to aorta)

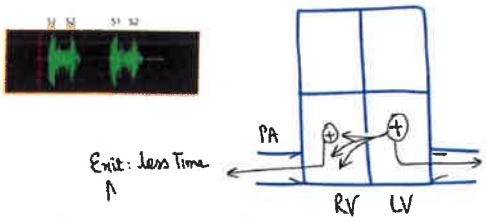
ON EXAMINATION	• S2: Wide spilt
	  ○ Exit of blood: less time
	  ○ Early closure of A2 (A2P2 gap: > 30 msec)
	• Pansystolic murmur



VENTRICULAR SEPTAL DEFECT (VSD)

00:40:07

GENERAL	• M/c congenital heart disease in infants
PATHOPHYSIOLOGY	• Shunt from left ventricle to right ventricle (during contraction of left ventricle) o Cause: Pressure of left ventricle > Pressure of right ventricle o Less blood pumped from left ventricle (to aorta)
ON EXAMINATION	• S2: Wide split o Exit of blood: Less time o Early closure of A2 (A2P2 gap: > 30 msec)
TYPES OF VSD	• Perimembranous VSD: M/c • Muscular VSD: Might exhibit spontaneous closure • Supracristal VSD : A/w valvular aortic regurgitation

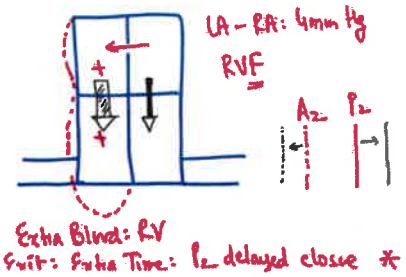


ATRIAL SEPTAL DEFECT (ASD)

00:43:47

→ Low-pressure shunt

- Pressure difference (between left atrium & right atrium): 4 mmHg
- Blood leaks from left to right atria → volume overloading of Right side of heart
- Causes right ventricular failure



→ On examination: Wide fixed split split S2

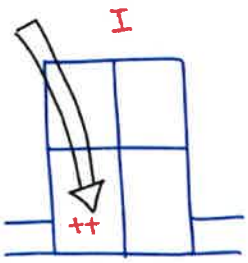
EXTRA BLOOD IN RIGHT VENTRICLE	LESS BLOOD IN LEFT VENTRICLE
• Exit of blood: Extra time • Delayed closure of P2	• Exit of blood: less time • Early closure of A2

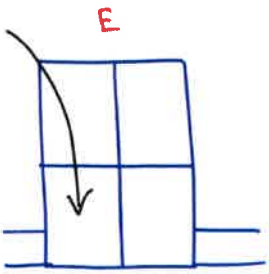
→ No shunt murmur heard

→ M/c age of presentation: 5 Years of age

- Rare adult presentation

	DURING INSPIRATION	DURING EXPIRATION
NORMAL	• ↑↑ blood pumped to heart • Exit of blood: ↑ time • Delayed closure of pulmonic valve	• ↓↓ blood pumped to heart • Exit of blood: ↓ time • Earlier closure of pulmonic valve

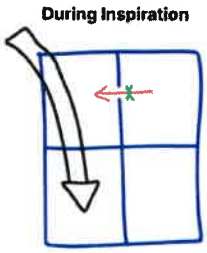




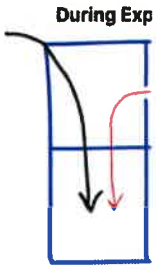
- Amount of blood in Right ventricle (in expiration & inspiration) → Variation
  - Physiological splitting of S2

ASD

- ↑↑ blood pumped to heart
- No pressure difference (between right & left atria)
  - No shunting

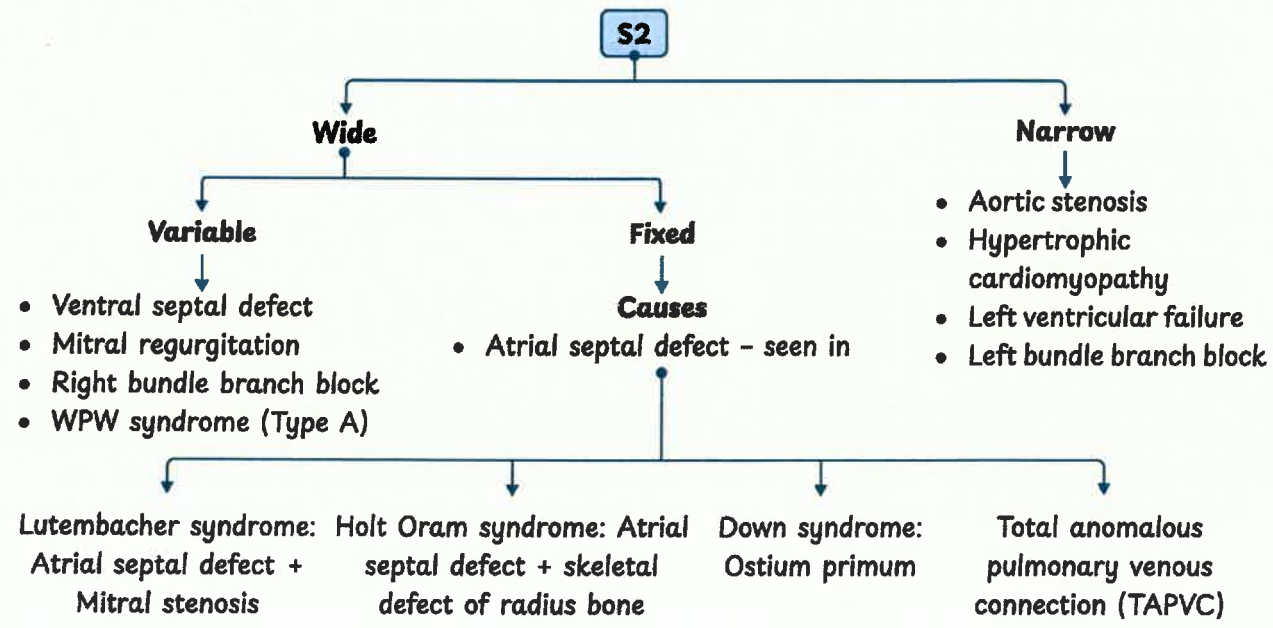


- ↓↓ blood pumped to heart
- Pressure difference (between right & left atria)
  - Shunting will occur (left atria → right atria)



- Amount of blood in Right ventricle (in expiration & inspiration) → same
  - No physiological splitting of S2
  - Wide fixed split S2 produced

Important Information



BUNDLE OF HIS

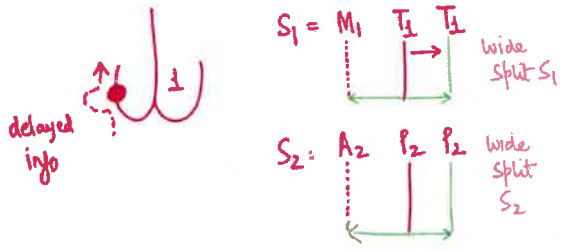
00:56:38

Responsible for septal activation

Current 2nd (secondarily) goes to right fascicle
- Tricuspid valve closes 2nd (S1 = M1T1)
- Pulmonic valve closes 2nd (S2 = A2P2)

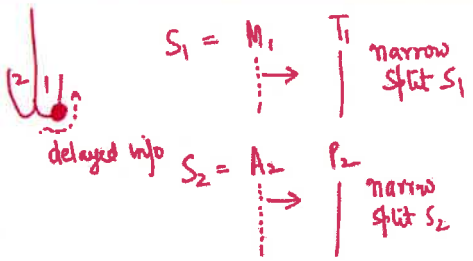
Current 1st (primarily) goes to left fascicle
- Mitral valve closes 1st (S1 = M1T1)
- Aortic valve closes 1st (S2 = A2P2)

RIGHT BUNDLE BRANCH BLOCK



- S1 = M1 T1
  - Delayed closure of T1 (due to delayed information)
  - Wide split S1
- S2 = A2 P2
  - Delayed closure of P2 (due to delayed information)
  - Wide split S2

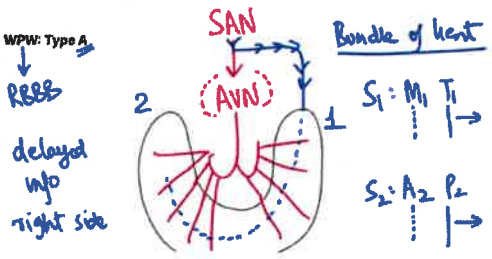
LEFT BUNDLE BRANCH BLOCK



- S1 = M1T1
  - Delayed closure of M1 (due to delayed information)
  - Narrow split S1
- S2 = A2P2
  - Delayed closure of A2 (due to delayed information)
  - Narrow split S2

WOLF PARKINSON WHITE (WPW) SYNDROME - TYPE A

- Short circuit of heart: 'Bundle of Kent'
  - Preferential conduction → Bypasses AV node
  - Current 1st passes through left ventricle (due to bundle of kent)
  - Delayed information to right side of heart
  - Behaves like Right bundle branch block
- S1 = M1T1
  - Delayed closure of M1 (due to delayed information)
  - Narrow split S1
- S2 = A2P2
  - Delayed closure of A2 (due to delayed information)
  - Narrow split S2



MCQ's

Q. Narrow Split S2 is heard in all of the following except?
a. Anterior wall MI
b. Hypertrophic cardiomyopathy
c. LBBB
d. WPW type A

Ans: (d)

Q. Inhaled Nitric oxide is used for the treatment of:
a. Labile hypertension
b. Pulmonary artery hypertension
c. Treatment-resistant migraine
d. Drug-resistant Parkinson's disease

Ans: (b)

Q. All of the following are correct about PDA except ?
a. Necrotising enterocolitis
b. B/L crackles on auscultation
c. Differential cyanosis on shunt reversal
d. Wide variable split S2

Ans: (d)

3. HEART SOUNDS PART 3

FIRST HEART SOUND: S1

LOUD S1

00:00:24

- Intensity α Speed of closure
- Pulse rate α 1/heart rate

LOUD S1 CAUSES

Tachycardia conditions
- Short PR interval
- Congestive heart failure (viral myocarditis)
- Thyrotoxicosis
- Pheochromocytoma
- Severe anemia

Physiological
- Pregnancy
- Child

Mitral stenosis/tricuspid stenosis -
↑ transvalvular gradient
↑ pressure in left atria → faster opening of mitral valve → faster elastic recoil → Loud S1

Important Information
- Calcified mitral stenosis - Soft S1
- Mitral stenosis-Delayed closure of Mitral valve →↑ Transit time
  - Narrow split S1 → Single S1 → Reverse split S1

SOFT S1

00:11:18

SOFT S1 CAUSES

- Bradycardia conditions -
  - Prolonged PR interval (1st degree heart block)
  - Hypothyroidism
  - SA node malfunction (sick sinus syndrome)
  - AV node malfunction (complete heart block)
  - Bundle of his malfunction (Mobitz type 2 heart block)

Obesity, emphysema

Mitral/Tricuspid regurgitation

Calcified severe mitral/tricuspid stenosis

THIRD AND FOURTH HEART SOUND

00:16:10

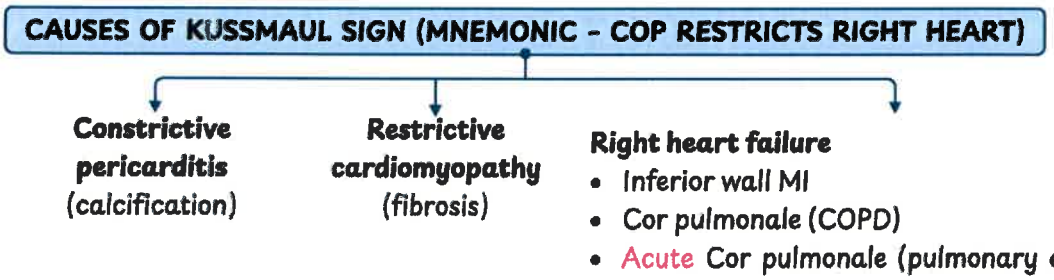
S3 (DILATED VENTRICLE)	S4 (OUTFLOW TRACT OBSTRUCTION - LVH → LAH (ATRIAL KICK)

• AKA: Ventricular gallop rhythm
• Rapid ventricular filling phase
• Seen in
  ○ Hypertensive crisis
  → Brain hemorrhage
  → Left ventricular failure
  ○ Dilated cardiomyopathy
  → Alcohol
  → Duchenne muscular dystrophy
  → Myocarditis sequelae
  ○ Congestive heart failure
  → Severe anemia
  → Viral myocarditis
  → PDA
  → VSD
  ○ Cor pulmonale
  → Right ventricular failure d/t non-cardiogenic cause- COPD
  ○ Chronic mitral regurgitation
	• AKA: Atrial gallop rhythm
	• Seen in
	  ○ Left-sided S4:
	  → Aortic stenosis
	  → Hypertrophic cardiomyopathy (subvalvular stenosis)
	  → Hypertension
	  ○ Right-sided S4:
	  → Pulmonic stenosis
	  → Pulmonary artery hypertension

JVP

00:26:10

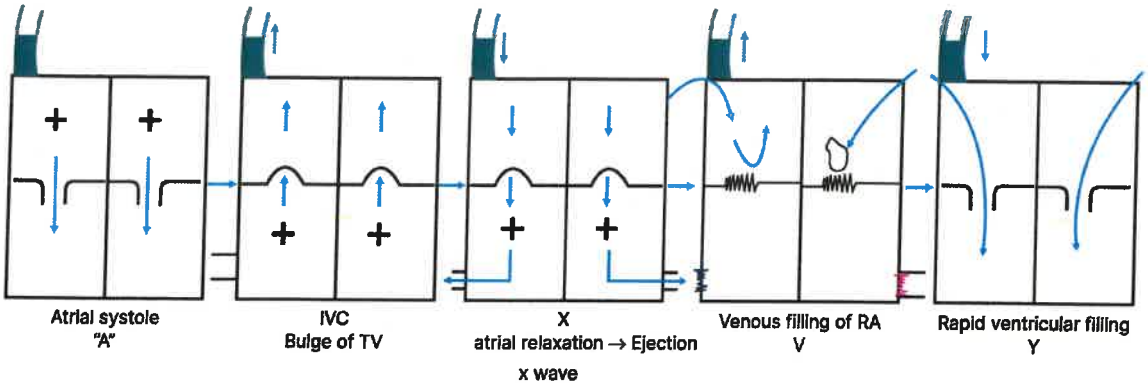
- Tells about the right atrium status
- Checked in internal jugular vein > external jugular vein (obese)
- Normal JVP: 5-8 cm of H_2O, measured from the Angle of Louis
- JVP falls on deep inspiration in a normal person
- Kussmaul's sign - Paradoxical rise on deep inspiration
- Non pulsatile ↑JVP - Cardiac tamponade



JVP WAVES

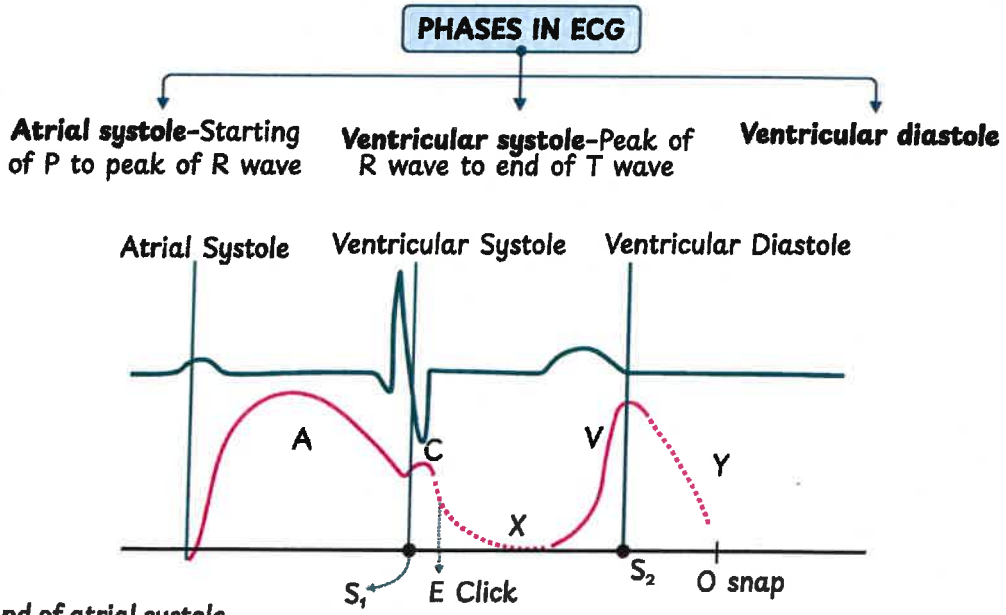
00:36:04

- a wave (↑) - Atrial systole
- c wave (↑) - Bulge of tricuspid valve (isovolumetric contraction)
- x-descent (↓)- Ejection phase/atrial relaxation
- v wave (↑)- Venous filling of right atrium
- y-descent (↓) - Rapid ventricular filling



Important Information

- Peak of v wave corresponds to S2

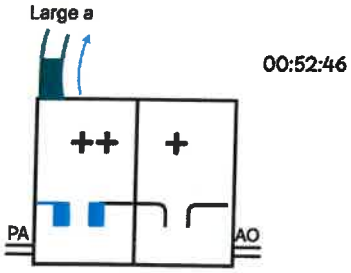


- S1 – At the end of atrial systole
- S2 - At the end of ventricular systole
- Ejection click – Transition between the c wave and x descent of JVP waves
  - Corresponds to the aortic and pulmonic valves opening
- After S2 – Opening snap (mitral/tricuspid opening)
- Iso volumetric contraction:
  - S1 at start
  - Ejection click at end

ABNORMALITIES OF JVP

LARGE a WAVE

- Tricuspid stenosis- Mid-diastolic murmur
- Pulmonic stenosis
- Pulmonary artery hypertension (scleroderma)
- TOF (subpulmonic stenosis)



- Long-standing mitral stenosis (right ventricular hypertrophy)
  - Long standing mitral stenosis → pulmonary edema and hypoxia → constriction of pulmonary artery → pulmonary artery hypertension

ABSENT a WAVE

- Atrial fibrillation (twitching) – Irregularly irregular pulse

CANON a WAVE/FROG SIGN

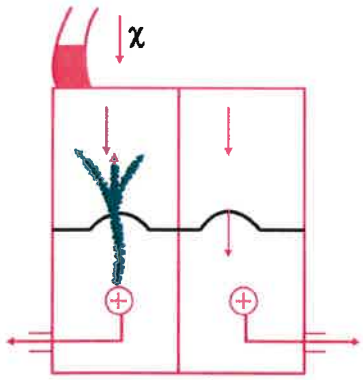
- AV dissociation

AV DISSOCIATION - CAUSES

- Ventricular tachycardia
- Junctional tachycardia- Retrograde P wave
- Complete heart block/3rd degree heart block/stokes adam syndrome

ABSENT x DESCENT

- Tricuspid regurgitation



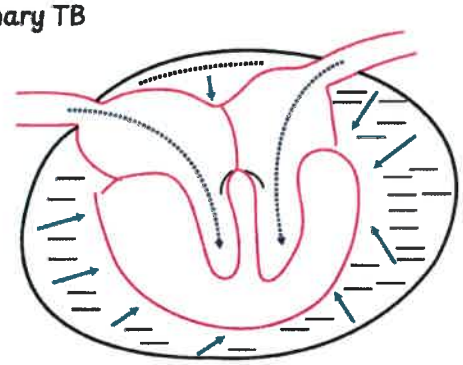
Absent x descent

cv WAVE /PROMINENT v WAVE

- Steep y descent – Severe tricuspid regurgitation

ABSENT y DESCENT- CARDIAC TAMPONADE 01:08:46

- Impaired ventricular filling (cardiac tamponade/pulseless electrical activity)
  - Cardiac tamponade is seen with Oat cell cancer lung, extrapulmonary TB
  - Cardiac tamponade – ↓ filling of ventricles, ↓ stroke volume, ↓ cardiac output, ↓ BP, pulse weak/ disappears
  - Pulsus Paradoxsus (pulse disappears during inspiration)
- Difference between inspiratory & expiratory SBP:
  - Normally: < 10mmHg
  - In cardiac tamponade: > 12mmHg


Cardiac tamponade